Unexpected outcome (positive or negative) including adverse drug reactions

Chronic inflammatory demyelinating polyradiculoneuropathy complicating anti TNF α therapy for chronic plaque psoriasisZahra Ahmed,¹ Robert Powell,² Gareth Llewelyn,² Alex Anstey³¹Dermatology Department, University Hospital Wales, Cardiff, UK;²Neurology Department, Royal Gwent Hospital, Newport, UK;³Dermatology Department, Royal Gwent Hospital, Newport, UK

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Summary

A 53-year-old woman with chronic plaque psoriasis treated with adalimumab (antitumour necrosis factor (anti TNF) α therapy) for 10 months presented with an 8 week history of hyperesthesia in a 'glove and stocking' distribution and clumsiness on walking. Nerve conduction studies confirmed the clinical diagnosis of a chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). She was admitted and treated with intravenous immunoglobulin and oral steroids and made an excellent recovery. To our knowledge, this is the first published report of CIDP associated with anti TNF α therapy given to treat psoriasis.

BACKGROUND

Antitumour necrosis factor (anti TNF) α drugs are now widely used by dermatologists, rheumatologists and gastroenterologists. It is important to recognise that although demyelinating peripheral neuropathy is a rare adverse event, it has considerable impact on patient morbidity. If recognised early, treatment with immunomodulatory drugs results in improvement.

Health professionals should be aware that neurological conditions may rarely complicate anti TNF α therapies.

CASE PRESENTATION

A 53-year-old woman with chronic plaque psoriasis treated with adalimumab for 10 months presented with an 8 week history of hyperesthesia in a 'glove and stocking' distribution and clumsiness on walking. She complained of heaviness of her feet and reduced dexterity of her hands. These symptoms progressed over this period to the extent that she was unable to walk without assistance. There were no respiratory, bulbar, bladder or bowel symptoms.

The patient had suffered from guillain-barré syndrome 29 years earlier, with complete recovery. Prior to starting biological therapy for psoriasis the risk of inducing a further demyelinating neuropathy was carefully considered by the prescribing dermatologist and a consultant neurologist. Following assessment of the published literature, it was concluded that this risk was small as there were no similar reports of previously fully resolved guillain-barré syndrome (GBS) that were precluded from having anti TNF α therapy. In addition, she had fully recovered from GBS with no reported relapses; the patient opted to proceed with treatment. She commenced treatment with efalizumab from november 2008 until April 2009 (cd11a inhibitor) which was later withdrawn due to drug safety concerns. In june 2009, she started anti TNF α therapy with adalimumab at a standard dose of 40 mg subcutaneous injection alternate weeks. She was on no concurrent medication.

Examination revealed both proximal and distal weakness of her upper and lower limbs with areflexia. She had marked distal loss of vibration and joint position sense, and a symmetrical reduction in pin prick sensation to the mid-thigh and wrist level.

INVESTIGATIONS

Blood tests for causes of peripheral neuropathy, spirometry and MRI brain/spine were all within normal limits. Nerve conduction studies confirmed the clinical diagnosis of a severe demyelinating neuropathy.

TREATMENT

Adalimumab therapy was stopped; the patient was admitted to hospital and treated with intravenous immunoglobulin (IVIG 0.4 g/kg/day for 5 days) with a good therapeutic response. Three further acute relapses over the subsequent 6 months required additional courses of IVIG, on each occasion with a good recovery. Oral prednisolone (0.5 mg/kg increased after 2 weeks 1 mg/kg) was introduced in order to prevent further relapses and withdrawn at a rate of 10 mg every 2 weeks. Twelve months later, the patient remains free of all treatment.

OUTCOME AND FOLLOW-UP

The patient is fully recovered and has been free from all neurological deficit for 7 months. She has been off prednisolone for 4 months.

A diagnosis of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) associated with adalimumab therapy was made and reported as an adverse drug event.

DISCUSSION

Anti TNF α drugs are now widely used by dermatologists, rheumatologists and gastroenterologists. Rare adverse

effects include local reactions, infections, congestive heart failure, malignancies, autoimmune and neurological events.¹ Central nervous system demyelination is well recognised,^{2 3} but peripheral nervous system involvement is rare. Reports of 34 patients with demyelinating neuropathies have been published to date including 15 cases of GBS.^{1 4} Reports of peripheral neuropathies with anti TNF α drugs are mainly limited to their use in rheumatoid arthritis patients, most of whom are more prone to the development of neuropathies. Three other cases of peripheral neuropathy have been reported in hidradenitis suppurativa, ankylosing spondylitis and Crohns disease.⁴

Clinical and neurophysiological findings can often be the same for GBS and CIDP. However, the distinction between GBS and CIDP is based on the time to maximal neurological deficit. For GBS this is 4 weeks; intermediate GBS is between 4 and 8 weeks, and >8 weeks is classified as CIDP. She achieved maximal neurological deficit at 9 weeks.

Generally, GBS has a 2–5% relapse rate but there are no data on the recurrence of GBS in association with anti TNF α therapy.⁵

Competing interests None.

Patient consent Obtained.

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Learning points

- ▶ Although demyelinating neuropathy is a rare adverse event of anti TNF α therapy, improvement usually occurs after stopping treatment and commencing immunomodulatory therapy.
- ▶ To our knowledge, this is the first published report of CIDP associated with anti TNF α therapy given to treat psoriasis.
- ▶ In this case, the relationship between CIDP and Guillain-Barré syndrome remains unclear.
- ▶ All health professionals should be aware that neurological conditions may rarely complicate anti TNF α therapies.

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